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IN A RESEARCH published by myself in the *Therapeutic Gazette*, in 1885, the observations of Chauvet, Carviler and several later investigators, that section of the spinal cord does not arrest, either in the upper or lower segment of the body, the choreic movements sometimes seen in diseased dogs, was confirmed by numerous experiments. It was further shown that whilst in advanced cases of canine chorea the ganglionic cells of the cord undergo distinct change, in the earlier stages of the disease, although the choreic movements may be well pronounced, no structural alterations can be detected in the nervous system. I also pointed out the close resemblance which exists between the chorea of the dog and the St. Vitus's dance of the child; and that since it is certain that the canine choreic movements originate in the spinal cord, it is most probable that the choreic movements in the human individual are often spinal. Since my research the matter has been considerably discussed by physiological clinicians, and it seems to me that the conclusion can, at this day, hardly be gainsaid, that whilst the movements which are called choreic may originate in the

cerebral cortex, spinal choreic movements occur in man as they do in other mammals. The assertion which has been made, that the mechanism of the choeric movement is altogether different in man and in the lower animal: that is, that it is altogether different in different species of animals, is too absurd for serious controversy. It would be just as well to waste time in discussing whether the mechanism by which voluntary movement is produced, or the method by which convulsions are brought about, differs in man and in the lower animals.

In the research of 1885, no direct answer was attempted to the question, "What is the immediate method in which choreic movements are produced;" or, in other words, why does the choreic movement replace the normal function of the part. It seems to me possible that the veterinarian and the human physician will agree that usually and probably always the spinal choreic movement is associated with absolute loss of power; a loss of power which varies in degree from mere failure of endurance to almost a complete paralysis.

The problem which is presented to us for study is the examination of the conjoint excesses of motion and loss of motor power. In thinking over this problem, it occurred to me that probably in chorea there is a condition of depression or exhaustion involving the two motor functions of the spinal cord, which I believe have been proven by the modern physiologist to exist, namely, the function of inhibition of motor discharge and the function of motor discharge. In other words, that the motor power of the spinal cells is weakened, but that the power of the inhibition apparatus, which controls motor discharge from these cells, is weakened to a greater extent than is the discharge power. The choreic movement may show itself simply in restlessness or fidgetyness, or it may reveal itself by excessive muscular contractions occurring under the impulse of the will; or in the severer cases there may be an irregular or a rythmic motor discharge, occurring entirely independently of the action of the will. According to the thought which I have, the quiet individual

is one whose inhibitory spinal apparatus is so powerful as to effectually control the motor cells of the cord; whilst in the restless individual inhibitory weakness reveals itself in the restlessness. As inhibitory weakness increases, restlessness increases, and the child becomes fidgety. When the stage of pronounced awkwardness and irregularity of movement is reached, the inhibitory spinal apparatus has so far lost control that it is no longer able to arrest muscular movements at the time when the necessities of the occasion demand that the motor discharge should cease, and so an excessive muscular response occurs to the will impulse because of the weakness of the apparatus which checks discharge from the cells concerned. In the most profound case of spinal chorea, human or animal, according to my thought, inhibition has become so far weakened that it has little or no power over the motor cells of the cord. As a further extension of the theory, the thought has occurred to me that it may be possible for a cell to revert to its original physiological type, just as it is possible for a part to revert to its original anatomical type. The original type of the nerve cell is that in which the cell gives rise to intermittent discharges, and therefore it is entirely supposable that the intermittent discharges of the choreic movement is due, first, to the natural tendency of the cell to discharge rhythmically, nerve force; and secondly, to the failure of that inhibition which in the normal cord prevents rhythymic discharge of nerve force.

A remarkable fact in connection with rhythmic choreic tremors is, that they may go on continuously without sensible fatigue; whereas, if a simple to and fro movement be made by the effort of the will, only a few moments will be required for exhaustion. The theory of the reversion of the cell to its original condition of rhythmic discharge, explains this fact; for we know that those cells which rhythmically discharge force do not tire. A normal heart continues to beat, it may be, through a century, without rest and without fatigue.

In regard to this theory of rhythmic discharge, I have

no evidence to offer the Society this evening; indeed, have not thought much over the matter. For putting it aside, I set myself to thinking whether or not it were possible, in any way, to throw light upon the condition of inhibition in the spinal cord of the choreic dog. My first series of experiments was directed to determining whether the motor cells of the spinal cord in the choreic dog are still capable of being inhibited. I have found that when division of the spinal cord is made low down, during etherization, the shock of the operation prevents a repetition of the movements for a considerable period after the recovery of consciousness. Thus, in one experiment, after division of the cord in the upper dorsal region, the movements did not reappear for over a quarter of an hour after complete recovery of consciouness. They first made their appearance in the hind legs, and afterwards in the front legs. The movements were finally much more violent in the hind legs than they had been before section; but all synchronism between the front and hind legs was completely set aside by the section.

In the second experiment the movements were rhythmical, and, as is often the case, were most marked in the opposing front and hind legs. They were, as in the first case, immediately stopped by ether. The cord was cut in the lower dorsal region, and the movements reoccurred in the front leg as soon as the effect of the anæsthetic had gone off, but did not come back in the hind legs for over an hour after the return of consciousness. It is plain to my mind that the shock of section of the cord has an effect upon these movements. In the first experiment, the front legs were nearest the seat of section, and recurrence of their movements was longest put off. In the second experiment, the section was practiced very low down, and the hind legs were inordinately influenced. In the choreic dog, as in the child, the movements can certainly be inhibited temporarily by the cerebrum.

Another experiment, of which I herewith submit the tracing,

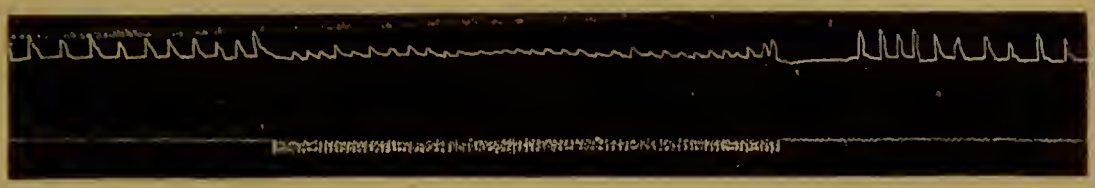

FIGURE 1. Widened space in lower line registered the application of faradic current to sciatic nerve.

confirms the fact that the function of inhibition in spinal chorea is still capable of being aroused. A complete section of the cord was made in a choreic dog, and although no evidence of pain was caused by very powerful faradization of the exposed sciatic nerve, the choreic movements were very markedly inhibited, as is shown in the photographic reproduction of the tracing made. The upper line represents the writing of the movements of the right leg, the lower line the time of application of the current. This experiment affords direct proof that there are inhibitory centres in the cord, or else that the ganglionic motor cells of the cord are directly inhibited by peripheral impulses.

In 1869, Dr. T. A. Chaperon proved that, in small doses, quinine caused in the frog a lessening in the reflex activity, which is relieved by high section of the spinal cord. The only explanation of this fact, which has been abundantly confirmed, is that the section has removed from the motor centres of the cord the influence of the stimulated inhibitory centres high up, and that therefore quinine is a stimulant to the inhibitory function of the cord. It is true that Dr. Sedgwick has combatted this theory, basing his opposition chiefly upon the fact which he discovered that atropine prevents the primary inhibition of reflexes by quinine.

It has, however, never seemed to me that the views of Sedgwick are entitled to much consideration; and the fact which he discovered is in accord with, and confirmatory of the theory of spinal inhibition. As first shown by Fraser, during recovery of the frog from atropine poisoning, there is a stage in which motor paralysis and

a reflex activity so excessive as to produce tetanus, coexist. The explanation of this as given by Fraser, has been proven to be incorrect by the experiments of Ringer and Murrell, whose views are summarized in the following passage taken from my Treatise on Therepeutics: "Drs. Ringer and Murrell believe that both the paralysis and the tetanus are due to a depressant action. The theory is that the normal cord has a power of resisting impulses received from the periphery, and especially of preventing their wide propagation among the spinal centres. During the first stage of the atropine poisoning it is supposed that the motor portions of the cord are so paralyzed as to be unable to form or propagate any motor impulse, and hence the general paralysis. Later on, however, the motor cells have so recovered themselves as to be able to generate impulses freely, although the resistive power of the cord is still in abeyance. Consequently a peripheral impulse plays as it were at will up and down the spinal cord, and instead of giving rise to a simple reflex action, gives origin to a series of reflex movements, involving all the muscles and constituting a tetanic convulsion."

It would seem, therefore, that atropine is a paralyzant; quinine, a stimulant of the inhibitory spinal function; and that one should antagonize the other is naturally to be expected.

The discovery of Sedgwick, that atropine brings back the reflexes arrested by quinine, is in verity a strong corroboration of the theory that the quinine acts upon the inhibitory apparatus. Holding, as I long have, that quinine is a stimulant and atropine a depressant of spinal inhibition, it occurred to me that if the choreic movements be due to weakness of the inhibitory centres of the cord, quinine should check the movements by strengthening the centres, whilst atropine should increase the movements by still further defining the centres.

I have made a number of experiments upon the action of quinine on the choreic movements in the dog, with results so concordant that it is certain that quinine, even

in small doses, does arrest the choreic movement. As evidence of this I offer to the Soceity the following tracing, which was made upon a revolving drum through a mechanical apparatus registering the movements of the paw of a choreic dog.

FIGURE 2. Lower line, seconds register. Upper line, register of choreic movement. Quinine injected at x.

With atropine I have had opportunity to make but a single experiment, not being able as yet to obtain another dog with chorea sufficiently pronounced to register itself well upon the drum. A tracing of this experiment I submit to you, and you will see that the results have been

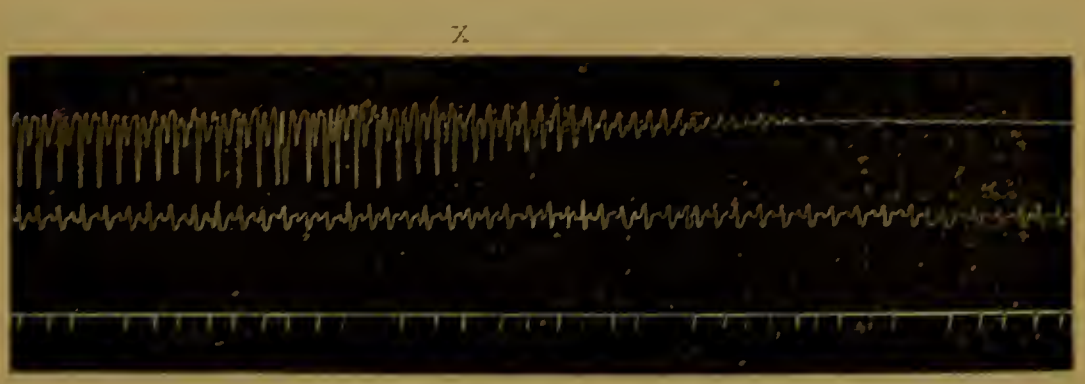

FIGURE 3. Lower line, second marker. Middle line, the register of choreic movement in the unpoisoned dog. The upper line shows same movements some minutes later after injection of atropine at position of cross. Quinine was intravenant injector.

remarkably decisive. The atropine quadrupled the extent of the choreic movements. You will notice, also, on

the tracing the registration of an immediate arrest of the movements produced by the injection of a small dose of quinine.[2]

Although in the choreic cord excessive movements occur as the result of the failure of inhibition, the partial, and finally it may be almost complete, paralysis which accompanies chorea shows that the motor cells themselves are functionally depressed, and indeed in my paper of 1885 it was shown that finally the motor cells, at least in canine chorea, undergo atrophic changes.

It is seemingly a priori, inevitable, that in chorea of a severe type the general muscular tone should be diminished, and that the reflexes, sharing in the general relaxation, should be less active, and in very severe cases even be abolished. The best explanation that has been offered of the phenomena of reinforcement of the knee jerks by voluntary movement, is that of the overflow; this widely accepted theory teaches that an influence destined primarily for a certain region of the cord has a tendency to spread itself throughout the cord. In the normal cord this overflow is more or less held in check by the inhibitory function of the spinal cord, and meets with resistance from the inhibitory function. A depression of the inhibitory function of the cord would therefore tend to diminish the resistance to the overflow, or in other words to increase the effects of the overflow. If the theory held in this paper in regard to the condition of the cord in the choreic movements be correct, we would expect to find in chorea, in the child, the knee-jerks lessened but the influence of reinforcement increased.

Knowing that my friend, Dr. Wharton Sinkler, had been engaged for some time in studying the knee-jerks in Sydenham's chorea, and knowing that his observations would be free from any possible influence by preconception, he having no knowledge of the inhibitory theory, herein propounded, I wrote asking him simply as to the

[2] Whilst this paper was going through the press a choreic dog having been secured it was found that atropine enormously stimulates the choreic movements.

condition of the knee-jerk in chorea, and received the following reply:

"I have examined the knee-jerks in the cases of chorea presenting themselves at the Infirmary for Nervous Diseases for several years. In the majority of cases the knee-jerk is diminished and in a few it is entirely absent except by reinforcement. Reinforcement in those cases where the knee-jerk is absent seems to produce excessive results, very strikingly shown in a patient at my clinic recently. This condition, I confess, I do not fully understand. One would rather expect exaggerated knee-jerks in chorea, as a result of the continuous muscular movement; supposing that these involuntary movements would have the effect of reinforcement. From this, one would infer that it requires a volitional act to cause reinforcement."

It is scarcely necessary to point out how these observations dovetail into the theory brought forward in this paper. As Dr. Sinkler says, the explanation of the reflex phenomena in chorea is extremely difficult with the older ideas of the origin of the choreic movement; that it is due to a simple excitement, of the motor cells. If, however, the choreic movement be of spinal origin, and if it be due really to a condition of depression of the spinal apparatus, no reinforcement is to be looked for from the choreic movement. It will be a matter of great interest to determine whether the choreic movement, so-called, which originate in the cerebrum, do or do not reinforce the knee-jerks. It seems to me a priori, therefore, that in cerebral chorea it will be found that the reflexes are reinforced by the motion.

As connected with the subject which I have been discussing, although I am not at present able to say what the exact relation is, I may call the attention of the Society to a discovery of Professor Reichert, of the University; namely, that it is possible to produce in the dog an artificial chorea. My friend, Prof. Chas. Richet, of the Ecole de Medecine, Paris, some years ago pointed out that if active artificial respiration be maintained, enormous

doses of strychnia injected into the veins of the mammal produce a complete paralysis. In studying this matter, Prof. Reichert has found that it is possible, by the use of the proper dose, to produce a condition in which neither the motor nor sensory nerves are able to respond to artificial stimulation, but in which the animal is violently choreic. The circulation is actively maintained but the reflexes are absolutely gone; so that violent irritation of the cornea elicits no response, and the most powerful faradization of the sciatic nerve produces no effect upon the circulation or upon the tributary muscles. Although sensory and nerve trunks are absolutely paralyzed, according to all ordinary tests, yet the motor nerve trunks still carry impulse from the centre when chorea occurs. I have found that quinine arrests these movements as it does the movements in the natural chorea of the dog. Atropine has not, in the various experiments which I have made, produced any distinct effect.

In concluding my remarks upon this subject, I want to distinctly say that the present communication has been made at this time partly on account of the interest of the subject, partly because I may have to wait months before I can get hold of another choreic dog, and partly because of the persistency of the secretary of the Society in his request for me to bring the matter before you. I do not hold myself in any way personally responsible for the correctness of the theory, and if further investigation shall disprove it, I will throw it aside as an old worn-out shoe.

The discovery of the power of quinine over the choreic movements has naturally led me to a trial of quinine as a remedy in chorea. I has, however, been only a few days since the present research was commenced. In the Clinic of the University Hospital, one case of marked chorea in a child presented itself. The child was cinchonized, and in two days the choreic movements had nearly stopped. As the case has not returned since it is probable that the relief continues.

Choreic dogs are frequently brought for treatment to

the Hospital of the Veterinary Department of the University of Pennsylvania, and speaking to Professor Pearson, he told me that at that time they had a very fine Irish setter suffering from chorea; that they had had a number of cases and so far failed to benefit any of them, and that he would try the quinine. Four grains of quinine have been given to the dog every three hours, at the present writing, for one week. The result has been almost complete disappearance of the choreic symptoms and extraordinary gain in power in the spinal cord. In the beginning of the administration the dog could scarcely stand; to-day he can walk some hundred of yards without difficulty. The future must, of course, decide whether these results are merely delusive coincidences, or whether quinine has curative power over chorea. Certainly, however, the quinine is worthy of trial.